Hydrocarbon aspiration in children and adolescents

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INTRODUCTION — Hydrocarbons are organic substances that contain carbon and hydrogen; they are liquid at room temperature [1]. All petroleum distillates (eg, kerosene, gasoline, mineral seal oils, and naphtha) are hydrocarbons; however, not all hydrocarbons are petroleum distillates. Hydrocarbons also often are mixed with agents that have systemic toxicity such as camphor, aniline dyes, heavy metals, and pesticides.

Ingestion of large quantities of hydrocarbons by children is unusual because hydrocarbons are foul-tasting. Aspiration of hydrocarbons by young children typically is an unintentional occurrence that can be prevented through safe packaging and storage. In contrast, hydrocarbon aspiration in teenagers usually occurs intentionally (eg, during inhalant abuse, or when attempting to siphon gasoline).

EPIDEMIOLOGY — Hydrocarbon ingestion accounted for approximately 2 percent of ingestions by children younger than six years of age in 2002 [2]. Between 1997 and 1999, an estimated 6400 children younger than five years of age were seen in hospital emergency departments for possible hydrocarbon aspiration after ingestion of household cleaning products [3]. Gasoline, lubricating oils, motor oils, mineral spirits, lighter fluid, naphtha, and kerosene were the most common exposures [4]. In 1998, unintentional ingestion of hydrocarbon resulted in the deaths of four children younger than 13 years of age; an additional 14 deaths were caused by intentional ingestion. Death from hydrocarbon aspiration usually is caused by respiratory failure.

CLASSES OF HYDROCARBONS — The four structural classes of hydrocarbons are:

- Aromatic hydrocarbons
- Halogenated hydrocarbons
- Terpene hydrocarbons
- Aliphatic hydrocarbons

Aromatic hydrocarbons are cyclic compounds containing a benzene ring (eg, benzene, toluene, and xylene). They are used primarily in solvents, glues, nail polish, paints, and paint removers [1]. Halogenated hydrocarbons are fluorinated, chlorinated, or brominated (eg, methylene chloride, chloroform, carbon tetrachloride, trichloroethylene, tetrachloroethylene). The terpenes include turpentine and pine oil. The aliphatic hydrocarbons are petroleum distillates. They are found in furniture polish, lamp oil, and lighter fluid [1].

HYDROCARBON TOXICITY — Hydrocarbons also can be classified according to their toxicity [1]:

- Nontoxic (unless complicated by gross aspiration) — Examples include asphalt, tars, mineral oil, liquid petrolatum, motor oil, axle grease, baby oil.
- Aspiration hazard — Clinical effects typically are limited to direct pulmonary damage and subsequent inflammation. Examples include turpentine, gasoline, kerosene, mineral seal oil (furniture polish), charcoal lighter fluid, cigarette lighter fluid, and mineral spirits.
- Systemic toxicity — Halogenated and aromatic hydrocarbons are absorbed readily through the gastrointestinal and/or respiratory systems. Systemic effects include cardiac arrhythmia and central nervous system (CNS) depression. In addition to halogenated and aromatic hydrocarbons, hydrocarbons that are combined with toxic additives (eg, organophosphates, heavy metals, camphor) also have systemic toxicity.

Determinants of toxicity — With the exception of aromatic and halogenated compounds, most...
hydrocarbons cause clinical toxicity only when aspirated or inhaled because they are poorly absorbed through the gastrointestinal tract. The aspiration hazard of the hydrocarbons is determined by three properties:

- Volatility — The ability to vaporize or to exist in a gaseous form
- Surface tension — The adherence (or cohesiveness) of molecules along a liquid surface; lower surface tension allows compounds to spread or “creep” over a larger area
- Viscosity — The resistance to flow through an orifice or the tendency of a compound to resist “stirring;” lower viscosity facilitates deeper penetration into the tracheobronchial tree

The systemic toxicity of hydrocarbons is determined by their volatility. Gases such as methane, ethane, propane, butane, and benzene are the most volatile. They cause asphyxia by replacing alveolar gas, are readily absorbed into the circulatory system, and cause CNS depression. However, they rarely cause pulmonary injury. Gasoline and naphtha also can cause direct CNS depression based upon their high volatility.

The aspiration hazard of hydrocarbons is inversely related to viscosity and surface tension and directly related to volatility. Thus, hydrocarbons with decreased viscosity, low surface tension, and high volatility are more likely to be aspirated and cause pulmonary injury. The low viscosity permits greater penetration into the distal airways while the low surface tension facilitates spread over a greater area. As an example, simple petroleum distillates (kerosene, gasoline, liquid furniture polish) are chiefly aspiration hazards. They have high potential to cause aspiration pneumonitis but rarely cause systemic symptoms.

**PATHOPHYSIOLOGY —** Hydrocarbon aspiration primarily affects the central nervous and respiratory systems. Volatile hydrocarbons are highly lipid soluble. They enter the circulation through the lungs and rapidly diffuse throughout the body and into the CNS [5,6] . Neurons, which have a high lipid content, are particularly susceptible to the solvent properties [7] . Manifestations in the CNS also occur secondary to severe pulmonary injury and hypoxia.

The respiratory system also is affected by direct injury. Low viscosity, low surface tension, and solvent properties of aspirated hydrocarbons together determine a compound’s ability to cause chemical pneumonitis [1] . The primary pathologic finding is severe necrotizing pneumonia. Other findings include direct destruction of the airway epithelium, alveolar septae, and pulmonary capillaries, as well as solubilization of the lipid surfactant layer. Secondary changes include atelectasis, interstitial inflammation, and hyaline membrane formation. The inflammatory response from chemical irritation generally causes temperature elevation, usually within hours of exposure.

**CLINICAL MANIFESTATIONS**

**Vital signs** — Between 30 and 60 percent of patients with hydrocarbon aspiration have fever at the time of presentation (38 to 40°C) [8] .

**Respiratory** — Pulmonary manifestations result from any degree of hydrocarbon aspiration, although their onset may be delayed for 12 to 24 hours. Immediate signs of aspiration include coughing, choking, gagging, and vomiting. Respiratory examination findings vary with the degree of pulmonary injury. Physical findings may include tachypnea, dyspnea, cyanosis, diminished resonance on percussion, suppressed or tubular breath sounds, and crackles. Displacement of alveolar gas by vaporized hydrocarbon may aggravate hypoxemia caused by inflammation and edema.

The major pulmonary complications of hydrocarbon aspiration include asphyxia, necrotizing chemical pneumonitis, lipid pneumonia, and hemorrhagic pulmonary edema, which quickly progresses to shock and respiratory arrest. Pneumothorax, subcutaneous emphysema of the chest wall, and pleural effusion, including empyema, also may occur. Secondary infection
with bacteria or viruses may occur. Pneumatoceles may develop in areas of extensive consolidation during the recovery period. (See “Spontaneous pneumothorax in children”).

**Radiographic findings** — The radiographic findings of hydrocarbon aspiration often occur before the development of physical findings. They may be seen within 20 minutes or as late as 24 hours after aspiration.

The initial findings are multiple, small, patchy densities with ill-defined margins. The lesions become larger and coalesce as the injury progresses. In some cases, the radiographic findings may be minimal at a few hours and then rapidly progress to extensive infiltrates. Emphysema or pneumothorax may develop. Radiographic abnormalities typically peak between two and eight hours after aspiration. The resolution of radiographic changes is gradual and lags behind clinical improvement, which usually occurs three to five days after aspiration. Pneumatoceles may develop in this latent period.

**Cardiovascular** — Cardiac arrhythmia may occur after inhalation. Solvent hydrocarbons can sensitize the myocardium to catecholamines, leading to fatal arrhythmia (“sudden sniffing death”).

**Gastrointestinal** — Ingestion of aliphatic hydrocarbons causes direct local irritation to the pharynx, esophagus, stomach, and small intestine, with edema and mucosal ulceration. Orogastric and intestinal irritation may be associated with nausea and hematemesis [1]. These effects usually are mild and rarely require treatment.

**Central nervous system** — Hydrocarbon ingestion or inhalation may have direct CNS effects, including somnolence, headache, ataxia, dizziness, blurred vision, weakness, fatigue, lethargy, stupor, seizures, and coma. In addition, hypoxia caused by hydrocarbon aspiration may cause secondary CNS depression, including drowsiness, tremors, or convulsions [9]. (See “Acute toxic-metabolic encephalopathy in children”).

**Hematologic** — Leukocytosis occurs early in the clinical course of hydrocarbon aspiration unrelated to pneumonitis and may last as long as one week [10]. Hemolysis, hemoglobinuria, and consumptive coagulopathy also may occur with significant ingestion [11].

**MANAGEMENT** — All children with hydrocarbon aspiration should be observed for at least six to eight hours in an emergency department setting. Chest radiographs should be obtained in all patients who have cough or any respiratory symptoms at presentation. Patients with normal initial radiographs should have them repeated four to six hours after ingestion.

**Decontamination** — The child’s clothing should be removed to prevent continued inhalation exposure. All patients should have their skin cleaned. The eyes should be flushed if any evidence of redness, tearing, or lid swelling is present.

As a general rule, decontamination of the gastrointestinal tract in children with hydrocarbon ingestion should avoid gastric emptying or lavage and induction of emesis because of the risk for aspiration during these procedures [12,13].

**Gastric emptying** — Gastric lavage may be indicated for certain hydrocarbon ingestions [1]:

- Those with systemic toxic effects (eg, halogenated and aromatic hydrocarbons)
- Petroleum distillates that contain toxic additives (eg, heavy metals or insecticides)
- Large volume of ingestion (eg, a suicide attempt)

The following measures, taken before gastric emptying, can minimize the risk of aspiration:

- Endotracheal intubation with a balloon-cuffed endotracheal tube
- Lateral decubitus or Trendelenburg positioning
- Pinching off the nasogastric or orogastric tube and withdrawing it quickly after the procedure is complete

**Pulmonary management** — The treatment of hydrocarbon pneumonitis is supportive. Endotracheal intubation is indicated in patients
with CNS depression or impaired ventilation [1,14]. Additional measures include oxygen, physiotherapy, and continuous positive airway pressure. Children who have hydrocarbon pneumonitis that is refractory to conventional supportive therapy may be candidates for receiving extracorporeal membrane oxygenation [15,16].

Bronchospasm should be treated with selective beta 2 agonists. Epinephrine and isoproterenol should be avoided because they can cause fatal ventricular dysrhythmia in the hydrocarbon-sensitized myocardium (see above).

Corticosteroids have no beneficial effect on the course of hydrocarbon aspiration [17,18]. Pneumatoceles rarely rupture and do not require treatment [19]. Pneumonitis caused by hydrocarbon aspiration should not be treated routinely with antibiotics unless signs of secondary infection, including the following, are present:

- Recurrence of fever after the first 48 hours [20]
- Increasing infiltrate in chest radiograph
- Leukocytosis

Disposition — Indications for immediate admission of children who have ingested or aspirated hydrocarbons include [21,22]:

- Symptomatic child with abnormal initial chest radiograph
- Patient with suicidal intent or massive ingestion
- Hypoxic or obtunded patient regardless of chest radiograph findings
- Patient with substantially abnormal chest radiograph

Indications for admission that become apparent during up to six hours of observation include:

- Child with mildly abnormal chest radiograph who develops symptoms during the observation period
- Child who develops symptoms related to toxic additives during the observation period (eg, heavy metal or organophosphate insecticide)
- Child with mild symptoms and normal chest radiograph who fails to improve during the observation period

All children for whom close follow-up cannot be established

Indications for discharge after six hours of observation include:

- Asymptomatic children with normal chest radiograph
- Asymptomatic children with mildly abnormal chest radiographs who do not develop symptoms during the observation period and who can receive adequate outpatient follow-up

PROGNOSIS — Although most children survive without complications or sequelae, some progress rapidly to respiratory failure and death. Systemic symptoms (eg, somnolence, convulsions, and coma) may dominate the course.

The prognosis is affected by the volume of ingestion or aspiration, the specific agent involved, and the adequacy of medical care. The typical clinical course averages two to five days. The mild CNS depression that is seen soon after ingestion seldom produces serious morbidity provided that pulmonary involvement does not occur.

The small airways are at greatest risk for long-term injury [23]. One study examined pulmonary function in 17 children eight to 14 years after hydrocarbon aspiration [24]. More than 80 percent had at least one pulmonary function abnormality. The clinical significance of these findings is uncertain.

SUMMARY — Patients with hydrocarbon ingestion should be observed for at least six hours because the symptoms and radiographic findings may be delayed.

- Thorough washing of contaminated skin and hair is an important part of therapy [13].
- Children who are symptomatic on presentation, who develop symptoms during the six-hour observation period, or who have ingested a particularly toxic agent (eg, furniture polish, organophosphate, heavy metal) should be admitted to the hospital.
- Children who remain asymptomatic during six hours of observation should continue to be
observed at home. Parents should be instructed to return if any respiratory symptoms occur.

- Pulmonary therapy is initiated based upon the development of symptoms. Cat cacholamines (eg, epinephrine and isoproteronol) should not be used to treat bronchospasm.
- Antibiotics should be used if the patient develops signs of secondary bacterial infection.
- Corticosteroids have no beneficial effect on the course of the illness as shown in double-blind controlled human studies [17,18].
- Pneumatoceles rarely rupture and do not require treatment.
- Parents should be reminded to keep cleaning fluids and kerosene out of the reach of children (eg, in locked cabinets, or out of the home).

REFERENCES